are likely to be least unco-operative, though the number of general practitioners to be relied on in a medical ward will probably be quite small. The whole scheme is therefore to be seen as a long-term project, to be implemented over may years. Gradually more and more centres may be affiliated to internal medical wards. Another hospital in Jerusalem has already taken on itself to run an additional district in the city, and the Beit Shemesh centre is going to expand during 1973. Other solutions not dealt with here have been tried by many general practitioners in Jerusalem.

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Medicine in Old Age

Disturbances of the Special Senses and Other Functions

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Sensory deprivation is now known to be a powerful weapon in causing perceptual disturbances.[1] Maintenance of contact with reality demands stimulation from the environment, and hence it is not surprising that old people who are deaf or blind or both appear to be confused—the links of communication to a sensorium with diminishing reserve have been severed. Social isolation and loss of independence are associated with loss of the special senses. These are compelling reasons to ensure that every old person can see and hear to his or her maximum capacity.

Impairment of hearing is often due to hard plugs of wax; removal by syringing after several days softening with Cerumol ear drops is a simple procedure. If deafness persists after removing the wax it is most likely to be due to either middle ear deafness or nerve deafness. A vibrating tuning fork is applied to the vertex of the skull in the midline and the patient is asked (in writing if necessary) to indicate whether the sound is heard in midline or in either ear (Weber's test); in middle ear deafness the sound is localized in the affected ear but in nerve deafness it is localized in the normal ear. In Rinne's test the vibrating tuning fork is applied to the mastoid process, the ipsilateral ear being closed. The patient is asked to indicate when the sound ceases and the vibrating tuning fork is then placed over the ipsilateral ear. In middle ear deafness nothing can be heard, but in nerve deafness the sound can still be heard. Most patients with middle ear deafness benefit from a hearing aid but in nerve deafness do not.

Otosclerosis is the commonest cause of middle ear deafness; rarer causes are Menière's disease, acute labyrinthitis, trauma, and therapy with streptomycin or ethacrynic acid. If the ipsilateral corneal reflex is absent then a full investigation for acoustic neuroma must be made.[2]

Efforts to preserve the sight must be no less vigourous, and mere chronological age is never a contraindication to cataract extraction. Sudden loss of vision in either eye demands sudden specialist attention lest the treatable go untreated. Acute glaucoma, arteritis, internal carotid artery occlusion, and detached retina all cause sudden deterioration in vision and are treatable. Detached retina is recognized when a well-demarcated area of the retina is seen moving like a sail with a well-defined margin; often there is associated haemorrhage into the vitreous. A pale optic disc with unfilled blood vessels is seen in carotid occlusion. A profuse haemorrhagic area indicates a venous thrombosis—usually limited to one venous segment. Primary optic atrophy is recognized by the appearance of a well-defined pale disc with the attenuation of all blood vessels; treatment of the cause such as syphilis can arrest the deterioration in vision. Unilateral or asymmetrical primary optic atrophy may result from a pituitary tumour or a sphenoidal wing meningioma.

Paget's disease of the skull may compress both optic nerves, with complete blindness, and the eighth nerve, with deafness. The value of thyrocalcitonin has so far not been proved in this condition, but when the sight and hearing are threatened it may offer the possibility of preserving them.[3] Elderly people may have primary optic atrophy from toxic exposure in earlier life such as tobacco, quinine, methyl alcohol, or inorganic arsenic about which nothing can be done. Chronic glaucoma may progress to give little more than tunnel vision, so that the patient may present with a history of bumping into objects. A hemianopia is invariably associated with a hemiplegia or some other evidence of a stroke.

Headache

Headache is not a feature of hypertension or space-occupying lesions in the elderly.

The headache of temporal arteritis represents an emergency because of the danger of sudden blindness[4] due to occlusion of the ophthalmic branch of the internal carotid artery. The patient with temporal arteritis may be vaguely unwell with limb pains and pyrexia; alternatively, the condition may present with sudden headache and vomiting. The E.S.R. is always raised. Examination of the temporal or occipital arteries will show a tender swollen artery which does not pulsate. The pathological process is one of gradual arterial occlusion and therefore warrants immediate therapy with steroids (60 mg, prednisolone daily). To avoid the dangers of long-term steroid therapy the affected artery

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should be biopsied because this minor operation seems to cause the condition to remit.

Cervical spondylosis is a common cause of occipital headache. Glaucoma may present with headache, vomiting, and photophobia; the increased intraoccular tension differentiates it from subarachnoid haemorrhage and meningitis. Headache due to arthritis of the temperomandibular joint is episodic and always worse at meal times. Sinusitis produces a headache which builds up to maximum intensity in the early afternoon and subsides towards evening; often there is an associated upper respiratory infection.

Tension headache is common and is recognized by the patient's description of "like a weight on top" or "like a band around the head"; headache associated with depression is recognized because usually there are other symptoms or signs of the condition.

Facial Pain

Trigeminal neuralgia or tic doulourex has its highest incidence in elderly women, being characterized by paroxysmal sharp pain in the second or third division of the trigeminal nerve. The pain begins suddenly and usually lasts about thirty seconds; eating, talking, touching the face, or a cold wind may all precipitate an attack. Spasm of the affected side of the face may occur with closing the eye and lachrymation. There are no abnormal physical signs between attacks. The waxing and waning nature of this affliction is seen in the middle aged but it wanes less often in the old patient, who may develop a depressive illness. The introduction of carbamazepine has changed the management of trigeminal neuralgia: rarely is alcohol injection or surgical resection of the trigeminal ganglion required nowadays. The side effects of giddiness and drowsiness make it imperative to introduce carbamazepine gradually, from 100 mg once daily to a maximum dosage of 200 mg thrice daily. This drug is an anticonvulsant with the usual toxic effects of agranulocytosis and aplastic anaemia.

Herpes Zoster

Herpes zoster, or shingles, has its highest incidence in older patients. It is an acute virus infection involving the first sensory neurone with a papulovesicular eruption in the corresponding dermatome. A few days of pain in the dermatome often precede the appearance of the segmental rash. Dusting powder and dry dressings on the eruption are usually all that is necessary but if there is secondary infection then systemic rather than local antibiotic therapy is indicated. Zoster may affect the ophthalmic division of the fifth cranial nerve, with corneal lesions and the risk of optic neuritis; when the sight is in jeopardy urgent systemic steroid therapy is required. Rarely zoster affects the geniculate ganglion, with the pain in the ear, lowermotor neurone facial paralysis, giddiness, deafness, and loss of taste in the anterior two-thirds of the tongue (Ramsay Hunt's syndrome). Most patients with herpes zoster recover without complications, the rash subsiding after seven days; however, a few develop postherpetic neuralgia, with intractable pain associated with depression. Many therapies have been tried for this condition but none seem any more effective than simple analgesics.

Vertigo

Vertigo may be a crippling malady. The commonest cause is postural hypotension. Normally the blood pressure remains constant irrespective of the posture; this independence of posture is maintained by baroreceptor reflexes operating from the aorta and the carotid sinuses. In old age the postural change requires a longer time for baroreceptor adjustment, especially when cerebrovascular disease is present[5]; hence rapid standing up can cause momentary dizziness. Old people should get out of bed in three stages;[6] they should first sit up, they should then dangle the legs over the side of the bed, and finally stand up. That an elderly person who feels giddy has postural hypotension can easily be confirmed by recording the blood pressure lying down, sitting, and standing. Clearly only one reading taken lying down can be deceptive. Postural hypotension is a manifestation of various diseases which interrupt or slow down the baroreceptor efferent pathway, and commonly it is seen in patients who are recovering from an illness with periods of immobility—for example, a stroke or congestive cardiac failure. In these instances gradual re-education of the baroreceptor reflexes occurs with increasing mobility.

Autonomic neuropathy as in diabetes mellitus, tabes dorsalis, or acute infective polyneuropathy are rare causes of postural hypotension. Drugs often cause troublesome postural hypotension; not surprisingly adrenergic-blockers such as bethanidine do so, but many other therapeutic agents also lower the blood pressure as a side effect—for example, diuretics, barbiturates, phenothiazines, and the tricyclic antidepressants.

Menières' Syndrome

Menière's syndrome may present for the first time in old age. The patient may have felt deaf for some time but then suddenly experiences tinnitus in one ear associated with a violent rotatory sensation, vomiting, profuse sweating, and prostration. There is rotatory nystagmus maximum to the side of the affected ear, and a tremor on the finger-nose test. Prochlorperazine is useful in the acute attack, but diuretic therapy to reduce the swollen endolymphatic system is disappointing. Should the attacks persist then a unilateral labyrinthectomy is indicated.

Giddiness associated with neck movement is rarely a symptom in patients with cervical spondylosis; here the atheromatous vertebral arteries are further narrowed by cervical osteophytes with resultant ischaemia of the brain stem. Other evidence of cervical spondylosis, such as wasting of the upper limb muscles or a spastic paraparesis, will be present.[7]

Drop Attacks

Old people often fall, but when a drop attack is the cause[8] there is a clear history that the patient fell without warning, did not lose consciousness, and had no amnesia. A patient may be standing or walking when the episode occurs; the patient rises quickly to her feet again; and there are no abnormal physical signs in the legs. Such attacks appear related to sudden failure of extensor tone in the legs but there is no evidence that vertebrobasilar insufficiency is the cause. There is no known treatment, but drop attacks wax and wane in frequency and often remit completely.

Subdural Haematoma

The highest incidence of subdural haematoma is in the elderly and in infants, with males predominating. Trauma is the commonest cause, but a history of this is often unobtainable, and when a good history is obtained the trauma may have been minimal. There is a recognized association with alcoholism. The diagnosis is easy if the symptoms im-

mediately follow an injury, but this is infrequently the case; more often there is an insidious onset weeks or months after the injury with fluctuating headache, drowsiness, and confusion.[9] Focal cerebral symptoms are unusual, but when present give a clinical picture resembling a stroke. Subdural haematoma are frequently bilateral. Electroencephalography is a disappointing test in the diagnosis of subdural haematoma, being less informative than the echo ultrasound; though angiography is hazardous, it is the best investigation, showing displacement of the cerebral arteries away from the inner table of the skull.

Epilepsy

The onset of epilepsy in the elderly usually follows a cerebrovascular accident; the seizure is grand mal in type and Jacksonian episodes are infrequent. A history of a recent stroke and the association of an upper motor neurone lesion in one or more limbs will be associated evidence. The fits can be controlled with primidone or phenytoin.

Uraemia, hypoglycaemia, and variable heart block may all provoke epileptiform seizures. In those patients with late onset epilepsy for which there is no obvious aetiology it is always a difficult problem to balance the points for and against a full neurological investigation; however, to do so one should be influenced by the patient's biological rather than chronological age.

The Dementias

There is wide individual variation in intellectual impairment in old age—a point appreciated fully when one sees a 95-year-old who is clear in the head attempting to converse with a confused 70-year-old.

For treatment and management it is mandatory to find the aetiology of the dementia, which is not a disease in its own right but a manifestation of a disease. Dementia secondary to vascular change is always associated with lateralizing neurological signs, such as a hemiplegia or bilateral extensor plantar responses. Other evidence of vascular disease is often found—for example, coronary artery disease or peripheral vascular disease.

Senile dementia or primary neuronal dementia is not associated with neurological signs or with vascular change. Prognostically the distinction is important. Patients with arteriosclerotic dementia may live indefinitely, but those with primary neuronal dementia rarely survive for more than 18 months after the diagnosis is made. Much evidence suggests that primary neuronal dementia has a genetic basis but few patients who carry the gene survive to the age when the effects are obvious.[10]

A treatable cause of dementia is normal pressure hydrocephalus. The aetiology and incidence of this are still undetermined, but these patients improve quickly after neurosurgical shunting of the ventricular cerebrospinal fluid into the venous system.[11 12]

Conclusion

Maintenance of cerebration, ambulation, sight, and hearing are essential for the quality of life and for an independent existence. The essentials depend more on the integrity of central nervous system than on any other system.

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Any Questions?

We publish below a selection of questions and answers of general interest

Dangers of Barbiturates for Asthmatics

In a recent leading article[1] it is stated that "even a small dose of a barbiturate or other narcotic drug can have serious consequences in patients with severe airways obstruction and hypercarbia." Is it wise, therefore, to give medicaments containing barbituaries to asthmatics?

When the author stated that . . . "even a small dose of a barbiturate or other narcotic drug can have serious consequences in patients with severe airways obstruction and hypercarbia . . ." he was primarily referring to cases of respiratory failure with a chronic bronchitis. Most asthmatic patients hyperventilate, have a low pCO_2, and do not fall into this category. In general, however, it is wise to avoid barbiturates, even in standard sedative doses in all patients with either chronic bronchitis with respiratory failure or with severe asthma. There is a recent evidence that barbiturates cause enzyme induction and therefore may reduce the effectiveness of corticosteroids in patients with asthma. This is an additional reason for not using barbiturates.

The top priority in drug management of asthma is to relieve airways obstruction. Once this has been achieved the patient will not require sedation. In fact, the dose of barbiturates in compound tablets is usually so low that it would be difficult to prove that they were harmful, but equally there is no evidence that such extremely low dosage is of any value. In general, it is probably better to use a selective β_2 stimulant such as salbutamol, terbutaline or orciprenaline in adequate therapeutic doses, rather than to use ephedrine in the very small dose contained in Franol tablets. In less severe asthma, it is well recognized that many patients apparently find one of the compound tablets, such as Franol, most suitable for them and under these circumstances the minute dose of barbiturate included is very unlikely to be important.

1 *British Medical Journal*, 1973, **2**, 320.